

Blood Gases:

Not as Complicated as They Seem

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What to do when someone hands you a blood gas:

First, **DON'T PANIC**. Analyzing a blood gas, even in the face of a complicated acid-base disorder, is well within the scope of even a beginning student. There is so much written about blood gases and acid-base disorders, however, and from so many perspectives, that it might look like a hopeless task. Never fear. With a few simple guidelines that you can keep on a card or in a pocket notebook, you can face the numbers without fear. The trick is to **start the right way**. There's lots of information on a blood gas. For simplicity's sake ignore it all except the pO_2 , pCO_2 , and pH; add to this the bicarbonate ("bicarb," CO_2) from an SMA panel (Chem-8, Basic Metabolic Panel) or set of electrolytes, and you're all set to evaluate oxygenation and diagnose acid-base status.

Arterial or Venous? VBGs rule!

The first question, before we even start delving deep into equations, and for that matter even before you order a gas, is whether to order an **arterial** or a **venous** gas. Yes, we talk about ABGs all the time. Yes, in community hospitals (and some teaching hospitals for that matter), the RTs will say "you want a what? A **venous** gas?" [aside to the other RT "do we do those?"]

Yes, "ABG" is a much more traditional term and what we always used to do.* But, hey, do **you** want someone to stick a needle in your artery if they can stick a needle in your vein and get the same exact information?

Back in the old days, we got ABGs mostly to find out how well oxygenated people were. Why? Because back before the days of pulse oximeters (you know, the "evil humour" days?), it was the only way. But now with pulse oximeters everywhere (I even have one in my car first-aid kit), that's much less of an argument. Indeed, we know that pulse oximetry, at least if you've got a good wave form, is quite accurate. Indeed, knowing the confusion about ABG results in sick-sick patients ("is this arterial or venous?" "maybe it's a mixed-venous gas... "get another one, OK?"), pulse oximetry is likely, in real, practical terms, **better** than an ABG. Well, this is true at least in the initial stages of evaluation, and as an emergency physician, that's my main interest—but it's also true when a patient on the floor suddenly decompensates.

Yes, if you have an indwelling arterial line in the ICU, and you're sure it's working OK, then following ABGs makes sense. But that's a tiny fraction of the patients who need their oxygenation checked.

And, it turns out that a **venous** blood gas can give you all the information you need to diagnose someone's acid-base status. OK, OK, there are a **few** exceptions, like someone in cardiac arrest or who is peri-arrest (that means either you just got them back or they're about to arrest). But for the majority of patients who are sick-sick but not circling the drain right on the edge, a **VBG** is just fine. It's faster and easier to draw, easier on the patient, and less likely to cause arterial bleeding or arterial clots. (Ever heard of the Allen test you're supposed to do before drawing an ABG at the wrist? That's to make sure the patient has two arteries to the hand—so if you wreck one, the hand doesn't turn black and fall off. Think about it.)

As you would expect, venous blood has much less oxygen than arterial blood. Normal arterial pO_2 (pAO_2) is about 80-100 mm; the corresponding oxygen saturation is anything greater than 95% saturation.

* Yes, yes, I know the footnote on page 3 talks about "SMA-6" and "evil humours" as being good examples of traditional terms I like to use. So I'm inconsistent.

Yes, there are few times when the pulse oximetry and the pO₂ don't match, primarily in carbon monoxide poisoning, so for carbon monoxide poisoning, a single ABG may make sense, at least for patients who are obviously sick. However, if the patient isn't sick and you just want to rule out CO poisoning, you can just get a venous CO level—they are just as good as arterial levels.¹

The **vast** majority of the time, as long as you can get a good pulse ox reading, you don't need an **arterial** blood gas. The table below is also available on a card at the end of this article, so you can keep it in your pocket:

SaO ₂	PaO ₂ @pH7.3	PaO ₂ @pH7.4	PaO ₂ @pH7.5
99	171	158	143
98	122	111	101
97	101	92	84
96	89	82	74
95	82	74	68
94	76	69	63
93	72	66	60
92	68	62	57
91	66	60	55
90	63	58	53
88	59	54	49
86	56	51	47
84	53	49	44
82	51	47	42
80	49	45	41
78	47	43	39
76	45	41	38
74	44	40	35
72	42	39	34

OK, so what are normal values for a **venous** blood gas?

The venous pO₂ is easy. Venous pO₂ is normally 40 mm Hg. This is the same as the normal arterial pCO₂, making it easy to remember.

The venous pCO₂ is also pretty easy. The venous pCO₂ is 5.7 mm Hg higher than arterial. For practical purposes, call it 6 mm Hg. Normal arterial pCO₂ is 40 (same as the venous pO₂ remember?), so normal venous pCO₂ is 46. Simple, really.

Normal pH for an arterial blood gas is 7.35-7.45, and normal for a venous gas is just a little more acidotic, as you'd expect: 7.32-7.42. So the venous pH is usually just 0.03 lower than the arterial. So take your venous pH and add 0.03 to it to get the equivalent in arterial-pH terms. But, really, 0.03 is a very small number, and probably not all that clinically significant, so if you just remember that the venous and arterial pH are about the same, that's good enough for government work.

Venous Blood Gas:

pH - 0.03 lower than arterial pH unless low-flow state (basically the same)

pO₂ - 40-50

pCO₂ - 5.7 higher than arterial pCO₂ (46 rather than 40)

So, if you're worried your patient has acidosis or carbon dioxide retention, just check a venous gas. If it's OK, then so's the patient's CO₂ level and acid-base status.²⁻⁵

Quick and Dirty

Looking to figure out someone's acid-base status in a hurry? Ignore all the verbiage in this handout except what's in the two chunks of boxed text below. (Something else that's on the card at the end so you can keep handy.)

Check the pH on the blood gas to tell whether the patient has an Acidemia (\downarrow pH) or Alkalemia (\uparrow pH) or EupHemia (normal pH).
Check the bicarb on the SMA-6 and the pCO ₂ on the gas.† \downarrow pH with \downarrow [HCO ₃ ⁻], or \uparrow pH with \uparrow [HCO ₃ ⁻], indicates a primary metabolic acidosis or alkalosis; \downarrow pH with \uparrow pCO ₂ , or \uparrow pH with \downarrow pCO ₂ , indicates a primary respiratory acidosis or alkalosis.
Check for the expected degree of compensation. (Empiric Equations Below)
For metabolic acidosis, check to see if it is an anion gap acidosis, a non-anion gap acidosis, or a mixed disorder. If all of the acidosis is due to anion-gap acids, then the increase in the anion gap should be the same as the decrease in the [HCO ₃ ⁻]. E.g., if the A.G. is 20 (normal 12), and the [HCO ₃ ⁻] is 19 (normal 27), then all of the acidosis (all of the \downarrow [HCO ₃ ⁻]) is due to the \uparrow A.G. from excess organic acids that form the anion gap. $AG = [Na^+] - ([Cl^-] + [HCO_3^-])$

There are about a zillion empiric equations that relate CO₂, pH, and bicarbonate. There are also complicated-looking graphs ("nomograms") that do essentially the same thing for you.

Just pick a couple of the simple equations to memorize or write down. Ignore all the rest unless someone forces you to look at them. My best picks are in the box below.

For metabolic acidosis, Winters' equation: $pCO_2 = 1.5([HCO_3^-]) + 8 \pm 2 = \text{last 2 digits of pH}$ for good compensation except: lactic acidosis may have overcompensation due to CNS effects.
For metabolic alkalosis or acidosis, with respiratory compensation: $pCO_2 = \text{last 2 digits of the pH} = [HCO_3^-] + 15$ (for [HCO ₃ ⁻] from 8 to 35)
For respiratory acidosis, every increase in pCO ₂ has an expected decrease in pH: $\uparrow pCO_2 \ 10 = \downarrow pH \ .08$ (acute) $\uparrow pCO_2 \ 10 = \downarrow pH \ .03$ (later, with metabolic compensation)
For respiratory alkalosis, each decrease in pCO ₂ has an expected increase in bicarbonate: $\downarrow pCO_2 \ 10 = \downarrow [HCO_3^-] \ 2$ (acute) $\downarrow pCO_2 \ 10 = \downarrow [HCO_3^-] \ 5$ (chronic/compensated)

And that's all you need to know about blood gas analysis.

Wellll, not really... you might want to know a bit more about what causes and maintains each of the four main

† "SMA-6" is the old name for "chem-6" or "basic metabolic panel." It means BUN, Cr, electrolytes; "SMA-7" adds a glucose. "SMA" is "Sequential Multi-Analyzer" which was the first commercial multiple-test lab chemistry machine. Yes, it's an old fashioned term, but hey, what's the matter with that? A sense of tradition is important, that's why I still use words like "monilia intertrigo" and "evil humours."

*None but respiratory alkalosis can have compensation all the way back to a normal pH. A normal pH with any other disorder indicates a second, superimposed, acid-base disorder. Also, note that SMA-6 autoanalyzer may not accurately determine very high HCO₃⁻; so the [HCO₃⁻] must be done manually by the lab.

abnormalities, and what to do about the problems you detect.

The remainder of this handout is a reference to the four primary acid-base disturbances, and the essential clinical features of each, also with postscripts on the A-a gradients and on blood gases and hypothermia. We start with the simple, and move to the more complex. The information in small print is somewhat esoteric, just some of my own notes from past lectures I've attended myself.

Respiratory Acidosis

First, remember the treatment for respiratory acidosis. It is not calculations, it is not drugs, it is **airway** and **breathing**. (Remember your ABCs?)

Second, respiratory acidosis tends to cause Cl^- loss and volume depletion by unknown mechanisms. (Source: Burton David Rose, *Clinical Physiology of Electrolyte and Acid-Base Disorders*, which is a just outstanding book.)

Respiratory Alkalosis

Causes:

- ❖ Salicylate overdose, especially in children (mixed with anion-gap metabolic acidosis, which predominates in later stages).
- ❖ Cirrhosis (\uparrow progesterone in blood?) and liver failure (\uparrow amines in blood?)
- ❖ Early stages of sepsis.
- ❖ Psychogenic hyperventilation.
- ❖ Pregnancy (\uparrow progesterone).
- ❖ Pontine lesions.
- ❖ Acute hypoxia (e.g., PE).

Respiratory alkalosis will \downarrow HCO_3^- slightly due to conversion to CO_2 .

Will have shift from pyruvate to lactate in blood, causing mild \uparrow anion gap compensatory acidosis.

Usually compensates totally over 2-3 days.

May cause asymptomatic \downarrow PO_4 .

Metabolic Acidosis

First question: is it adequately compensated?

Use one of the two formulas in the box above.

Second question: is it an Anion Gap or a Non-anion-gap (hyperchloremic) metabolic acidosis?

- ❖ $[\text{Na}^+] - ([\text{Cl}^-] + [\text{HCO}_3^-]) >> 12$ is anion gap acidosis
- ❖ $[\text{Na}^+] - ([\text{Cl}^-] + [\text{HCO}_3^-]) = 12$ is hyperchloremic (non-anion gap) acidosis
- ❖ \uparrow in anion gap should be same as \downarrow in HCO_3^- , or has BOTH anion-gap and non-anion-gap metabolic acidoses.

High Anion Gap Metabolic Acidoses

Mnemonic-- KUSMAL:

- K- ketoacidosis
- U- uremia
- S- salicylates

M- methanol (and ethylene glycol, paraldehyde)

A- alcohol (EtOH)

L- lactic acidosis

Normal Anion Gap (Hyperchloremic) Metabolic Acidoses

divided into high K^+ vs. low K^+

a. high K^+

1. hyperal and increased amino acid catabolism
2. post-hypocapnic (it takes a while for the HCO_3^- to rise)
3. dilutional
4. hypoaldo (either from low renin or adrenal dysfunction)
5. NH_4Cl , $CaCl_2$, lysine, or arginine (effectively adds HCl)

b. low K^+

1. GI: diarrhea, fistulas (K^+ loss and high aldo)
2. GU: surgical ureteral conduits
3. RTA: Renal Tubular Acidosis. Complex subject best left to outside reading or a nephrology rotation.

Metabolic Alkalosis

Metabolic alkalosis is a complex disorder, and understanding of metabolic alkaloses has generally been an arcane art, whose practitioners are suspected of pacts with the devil. However, it need not be quite so complex. This presentation provides three different views of metabolic alkalosis: **Pathophysiology**, **Classification**, and **Treatment**.

Pathophysiology of Metabolic Alkalosis

PRIMARY EVENT

The primary event is either loss of H^+ or gain of HCO_3^- . Examples: upper GI losses from vomiting or NG suction, H^+ loss from the kidney. Or, H^+ may move into the cells because of a low serum K^+ . Alkalosis may also result from addition of HCO_3^- or (purportedly) "contraction" of fluid volume around a fixed amount of HCO_3^- .

MAINTENANCE

Alkalosis will quickly be corrected by compensatory mechanisms unless some factor is acting to maintain the alkalosis. Several factors may maintain an alkalosis, all by decreasing HCO_3^- excretion (italics below):

- A high aldosterone state, either primary or secondary[§]
- $\downarrow K^+$ or $\uparrow Cl^-$
- The renal T_m for HCO_3^- may be reset \uparrow by all of the above, and by \downarrow volume.**
- Non-resorbable anions (e.g., penicillin, carbenicillin) take HCO_3^- with them.

[§]SECONDARY HYPERALDO may be due to: \downarrow volume, or \downarrow effective volume (e.g. in cirrhosis, CHF, nephrotic syndrome); or $\downarrow Cl^-$ (perhaps the effector of decreased volume); both cause secondary \uparrow aldo (which causes sodium avidity in the distal tubule).

PRIMARY HYPERALDO may be due to: tumor, licorice (glycyrrhizic acid in licorice is similar to aldo), or Bartter's syndrome: congenital hypertrophy of juxtaglomerular apparatus with \uparrow renin and inability to reabsorb Cl^- .

High aldo (along with $\uparrow Na^+$ delivery to the distal aldo-sensitive areas of the tubule from diuretics or $\uparrow HCO_3^-$ over what the proximal tubule can absorb) causes $\uparrow Na^+$ reabsorption and obligate K^+ or H^+ excretion. Attempts to retain Na^+/HCO_3^- by the distal tubule lead to secretion of H^+ to permit recovery of the HCO_3^- ; otherwise, a large amount of Na^+ would be lost along with the HCO_3^- , resulting in volume depletion.

**Mechanism: increased H^+ in renal tubule cells (because of K^+ efflux in K^+) causes $\uparrow H^+$ excretion.

- $\uparrow p\text{CO}_2$ secondary to alkalosis causes a TERTIARY renal HCO_3^- retention (up to 40% of the $\uparrow \text{HCO}_3^-$ in metabolic alkalosis may be secondary to this).^{††}
- Renal failure with decreased HCO_3^- filtration (rare)
- A metabolic alkalosis (but not alkalemia) may be a compensatory loss of H^+ due to primary respiratory or metabolic acidosis. (Caused by $\uparrow \text{H}^+$ in renal tubule cells.)

CLASSIFICATION OF METABOLIC ALKALOSIS:

This section is in smaller print because it's more complicated than the rest and probably not worth reading until you actually have a patient with metabolic alkalosis in front of you. Three different views of metabolic alkalosis are presented. Each classifies all the alkaloses in a different way. No one classification is clearly superior.

Classification #1: Chloride-responsive vs. Non-chloride responsive Metabolic Alkalosis

a. Chloride Responsive Metabolic Alkalosis

Urinary $\text{Cl}^- < 20$ mEq/l means chloride-responsive, which will respond to NaCl. Most alkaloses are in this category and are associated with normal or low blood volumes:

1. Gastric fluid loss
2. Diuretics
3. Posthypercapnic
4. Diarrheal losses of Cl^- (congenital or villous adenoma)

b. Chloride Unresponsive Metabolic Alkalosis

Urinary $\text{Cl}^- > 20$ mEq/l means not chloride-responsive. These alkaloses are rare, and are associated with normal to increased plasma volume. The problem is volume expansion secondary to hyperaldo., severe $\downarrow \text{K}^+$, or severe $\uparrow \text{Cl}^-$, + leading to HCO_3^- loss; give plenty of K^+ , treat with large doses of Aldactone (spironolactone) to inhibit the aldo effect. Causes:

1. Conn's disease (primary hyperaldo)
2. Cushing's syndrome (cortisol has some mineralocorticoid effect)
3. Congenital adrenal hyperplasia
4. Bartter's syndrome (congenital hypertrophy of juxtaglomerular apparatus with \uparrow renin and inability to reabsorb Cl^-)
5. true licorice ingestion (glycyrrhizic acid in licorice is similar to aldo)
6. ? profound K^+ depletion with \downarrow GFR

c. Unclassified Metabolic Alkalosis

1. alkali administration
2. milk-alkali syndrome
3. glucose ingestion after starvation (unknown mechanism)
4. blood transfusion (citrate)
5. large doses of penicillin or carbenicillin (cause increased excretion of K^+ and H^+ due to large filtered load of - ions)

Classification #2: Metabolic Alkalosis by Root Causes

- a. Upper GI H^+ losses (vomiting or NG suction)
- b. Renal H^+ losses (e.g. diuretics, hyperaldo)
 1. Diuretics ("contraction"); actually, diuretics probably cause metabolic alkalosis by mechanisms described under "pathophysiology" rather than by contraction around the HCO_3^-
 2. Primary mineralocorticoid excess
 3. K^+ (severe) with \downarrow GFR
- c. Colonic H^+ losses (diarrhea or villous adenoma or laxative abuse with secondary renal dysfunction?)
- d. Alkali ingestion (IV or PO bicarb or CaCO_3) (rare)
- e. Posthypercapnic: after CO_2 normalized, takes a while for the compensatory $\downarrow \text{HCO}_3^-$ to normalize.
- f. Hypoparathyroidism: PTH causes HCO_3^- excretion, lack $>$ retention.

Classification #3 of Metabolic Alkaloses: Anion Gap vs. non-Anion Gap

a. High Anion Gap Metabolic Alkaloses

1. $\uparrow +$ charges on plasma proteins from \uparrow pH
2. increased lactate production
3. decreased lactate metabolism (e.g. in liver failure with hyperalimentation)

^{††}"Metabolic alkalosis", Harrington, John T., Kidney International, 26(1984) 88-97.

b. Normal Anion Gap Metabolic Alkaloses

1. ↑ Ca⁺⁺
2. ↑ Mg⁺⁺
3. paraproteins
4. lithium

Treatment of Metabolic Alkalosis**PH < 7.6, ASYMPTOMATIC (CHLORIDE-RESPONSIVE ASSUMED)**

- ❖ **volume depleted:** give IV-NS and K⁺ (base amount of K⁺ on urine Cl⁻); see below.
- ❖ **edematous but intravascularly depleted** (CHF, nephrosis, cirrhosis): give albumin to ↑ intravascular volume if appropriate, replete K⁺ aggressively, and consider Diamox (see below)
- ❖ **volume expanded (rare):** give KCl po or IV

PH > 7.6, SYMPTOMATIC (CHLORIDE-RESPONSIVE ASSUMED)

- ❖ monitored bed
- ❖ NH₄Cl or HCl IV
 - 20-40 mEq/hr of 0.1 N or 0.15 N HCl through central line (NH₄Cl can be given PO, but must be avoided in cirrhosis or renal failure because of NH₃ buildup)
 - 100 mEq H⁺ should ↓ HCO₃⁻ by 7 mEq/L
 - follow hourly pH, pCO₂, HCO₃⁻
 - stop when pH < 7.6 and patient asymptomatic
- ❖ Acetazolamide (Diamox)
 - watch for ↓ K⁺; must supplement with massive amounts KCl
 - 250 mg IV Q8H
 - stop when pH < 7.6 and patient asymptomatic

NON-CHLORIDE-RESPONSIVE:

Urinary Cl⁻ > 20 mEq/l means not chloride-responsive (rare), so problem is probably primary hyperaldo, or severe lack of K⁺ leading to HCO₃⁻ loss; give plenty of K⁺, and give large doses of Aldactone (spironolactone) to block aldo receptors.

Further Reading

If you are still puzzled by acid-base balance, and not too nauseated by the dense information above, you should pick up a good book on the topic. There's a book by Stryer that is very well-thought-of, but my personal favorite is *Clinical Physiology of Acid Base and Electrolyte Disorders* by Burton David Rose. If you can't find it in the bookstore, get it from www.amazon.com.

I hope you find this little handout useful. It's just cobbled together from my own notes, and is pretty rough, but remember, it's at least worth what you're paying for it (nothing).



Appendix I: Oxygenation: the A-a gradient

Evaluating the pO₂ on room air is easy: the lab usually prints out normal values for you, and even puts a little asterisk next to low values. But what if the patient's pO₂ is low normal, but the pCO₂ is below normal. Could this mean the patient is having to work hard to keep the pO₂ up, causing a low pCO₂? Could this mean the patient has a big pulmonary embolism or a bad pneumonia, despite a "normal" pO₂? You betcha. ##

Is there any scientific way to factor the pCO₂ into your figuring? Sure enough. This calculation has the intimidating name of the **Aa Gradient**. And, deriving it from first principles can be difficult. But using the formula is incredibly simple. Just keep it written down somewhere so you can find it when you need it.

A-a Gradient (Alveolar-arterial Oxygen Gradient)

Use: To quantify ABG results to estimate the average gradient across the alveolar membrane. An increased A-a gradient is seen in pulmonary embolism, pneumonias, ARDS, etc.

Normal A-a gradient:

Age	Upper Limit of Normal
20	17
30	21
40	24
50	27
60	31
70	34
80	38

From: Morris A, Kamer R, Crapo R, et al. Clinical pulmonary function: a manual of uniform laboratory procedures, 2E. Salt Lake City: InterMountain Thoracic Society, 1984:45. Quoted in Emergency Medicine Reports, 12/14/92.

Normal values increase with age, and smoking or occupational pneumoconiosis will also increase the A-a gradient.

Equation: A-a gradient = (713 (FIO₂) - 1.2 (pCO₂)) - (pO₂)

For room air: A-a gradient is: 150 - 1.2(pCO₂) - pO₂

Derivation:

(pAlveolarO₂) = (Fractional Inspired O₂) * (Atmospheric Pressure - partial pressure of water in saturated air) - (pAlveolar CO₂/R) -- (where R is the respiratory coefficient, the amount of CO₂ produced per molecule of O₂ used) or:

One note: if trying to diagnose pulmonary embolism in a patient with chest pain, syncope, or unexplained tachycardia, don't use the A-a gradient, or a pO₂, or even a pulse oximeter reading to help you decide if it's a PE. It just doesn't help. Yes, by all means check the patient's oxygenation to see how sick the patient is and whether the patient needs oxygen. But the majority of people with a PE will not have low oxygenation. Indeed, it's even said (correctly) that, if you have two young healthy women with pleuritic chest pain, and one has a normal pulse ox and the other has a low pulse ox, the one with the normal pulse ox is more likely to have a PE. True. The one with the low pulse is more likely to have a pneumonia.

$pAO_2 = (FIO_2(760-47)) - (pACO_2/R)$ or,

since rapid diffusion of CO_2 across the alveolar membrane means that $pACO_2$ and $paCO_2$ are the same:

$pAO_2 = 713(FIO_2) - 1.2(paCO_2)$

therefore: $A-a O_2 = (713(FIO_2) - 1.2(pCO_2)) - pO_2$

Appendix II: Hypothermia and blood gases

You may hear about "correcting blood gases for hypothermia." The blood gas blood is warmed to room temperature when the syringe is injected into the ABG machine. Therefore, the lab wants to know if the patient is hypothermic, so they can "correct" the numbers. Lab people want to "correct" the numbers so that they give you exactly correct numbers, reflecting exactly what the pH and pCO_2 are in the patient's blood. Despite these good intentions of the lab people, **LIE TO THEM**. If they ask if the patient is hypothermic, say no. If someone is taking the gas to the lab, and might tell the lab the patient is hypothermic, use duct tape over the person's mouth before sending him or her to the lab.

Why? One very important reason.

You've invested considerable time and energy in learning about room temperature blood gases. But, do you have any idea what a normal pH or pCO_2 is at $30^\circ C$? At $25^\circ C$? I don't, and hypothermia is one of my specialties. But it turns out that if you warm the blood gas to a normal temperature, **and don't correct for hypothermia**, then the numbers you get compare almost perfectly with a room-temperature gas. For example, the patient is hypothermic to $25^\circ C$. You get an **uncorrected** pH of 7.4. This means the patient is not acidotic (acidemic), and not alkalotic (alkalemic). If you got a "corrected" gas, you'd have to go look up in a table what the normal pH is for a core temperature of $25^\circ C$. *The normal pH at $25^\circ C$ is **not** 7.4. Same thing for pCO_2 . The normal pCO_2 at $25^\circ C$ is **not** 40.*

Got it?

There is one exception, though. Correcting for temperature is appropriate when you're considering pO_2 on an **arterial** gas. Here's the trick I use: send the gas up without any mention of hypothermia, then use the pCO_2 and pH to figure out the patient's acid-base status. Then call the lab and say "oh, by the way, the patient's core temperature is $25^\circ C$, so you could you tell me the corrected pO_2 ?" And when they try to give you the corrected pH and pCO_2 , just put your fingers in your ears.

Well, now you know why you keep the lab people in the dark about your patient's temperature: to keep them from confusing you, and to let you use your room-temp acid-base knowledge to properly manage your patient.

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Blood Gas/Acid Base Reference Card

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Check the pH on the blood gas to tell whether the patient has an Acidemia (\downarrow pH) or Alkalemia (\uparrow pH) or EupHemia (normal pH).

Check the bicarb on the SMA-6 and the pCO₂ on the gas. \downarrow pH with \downarrow [HCO₃⁻], or \uparrow pH with \uparrow [HCO₃⁻], indicates a primary metabolic acidosis or alkalosis; \downarrow pH with \uparrow pCO₂, or \uparrow pH with \downarrow pCO₂, indicates a primary respiratory acidosis or alkalosis.

Check for the expected degree of compensation. (Empiric Equations Below)

For metabolic acidosis, check to see if it is an anion gap acidosis, a non-anion gap acidosis, or a mixed disorder. If all of the acidosis is due to anion-gap acids, then the increase in the anion gap should be the same as the decrease in the [HCO₃⁻]. E.g., if the A.G. is 20 (normal 12), and the [HCO₃⁻] is 19 (normal 27), then all of the acidosis (all of the \downarrow [HCO₃⁻]) is due to the \uparrow A.G. from excess organic acids that form the anion gap. $AG = [Na^+] - ([Cl^-] + [HCO_3^-])$

For metabolic acidosis, Winters' equation:

$$pCO_2 = 1.5([HCO_3^-]) + 8 \pm 2 = \text{last 2 digits of pH}$$

for good compensation except:

lactic acidosis may have overcompensation due to CNS effects.

For metabolic alkalosis or acidosis, with respiratory compensation:

$$pCO_2 = \text{last 2 digits of the pH} = [HCO_3^-] + 15$$

(for [HCO₃⁻] from 8 to 35)

For respiratory acidosis, every increase in pCO₂ has an expected decrease in pH:

$$\uparrow pCO_2 \ 10 = \downarrow pH \ .08 \ (\text{acute})$$

$$\uparrow pCO_2 \ 10 = \downarrow pH \ .03 \ (\text{later, with metabolic compensation})$$

For respiratory alkalosis, each decrease in pCO₂ has an expected increase in bicarbonate:

$$\downarrow pCO_2 \ 10 = \downarrow [HCO_3^-] \ 2 \ (\text{acute})$$

$$\downarrow pCO_2 \ 10 = \downarrow [HCO_3^-] \ 5 \ (\text{chronic/compensated})$$

Normal Venous Blood Gas values:

pH: 7.32-7.42 (0.03 less than arterial: 7.35-7.45)

pCO₂: 46 (6 more than arterial: 40)

pO₂: 40-50

Blood Gas/Acid Base Reference Card
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SaO ₂	PaO ₂ @pH 7.3	PaO ₂ @pH 7.4	PaO ₂ @pH 7.5
99	171	158	143
98	122	111	101
97	101	92	84
96	89	82	74
95	82	74	68
94	76	69	63
93	72	66	60
92	68	62	57
91	66	60	55
90	63	58	53
88	59	54	49
86	56	51	47
84	53	49	44
82	51	47	42
80	49	45	41
78	47	43	39
76	45	41	38
74	44	40	35
72	42	39	34

High Anion Gap Metabolic Acidoses Mnemonic

KUSMAL:

- K- ketoacidosis
- U- uremia
- S- salicylates
- M- methanol (and ethylene glycol, paraldehyde)
- A- alcohol (EtOH)
- L- lactic acidosis

Normal Anion Gap (Hyperchloremic) Metabolic Acidoses
 divided into high K⁺ vs. low K⁺

a. high K⁺

1. hyperal and increased amino acid catabolism
2. post-hypocapnic (it takes a while for the HCO₃⁻ to rise)
3. dilutional
4. hypoaldo (either low renin or adrenal dysfunction)
5. NH₄Cl, CaCl₂, lysine, arginine (~= adds HCl)

b. low K⁺

1. GI: diarrhea, fistulas (K⁺ loss and high aldo)
2. GU: surgical ureteral conduits
3. RTA: Renal Tubular Acidosis. Complex.